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Review Article

Association of sleep and sleep disorders with glaucoma

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ABSTRACT

The hormone melatonin plays a role in our natural sleep-wake cycle. Natural levels of melatonin in the blood are highest at night. Aqueous flow has a distinctive circadian rhythm, being lower at night than during the day. IOP fluctuates in a rhythmical diurnal pattern and this fluctuation is greater in eyes with glaucoma. Sleep apnoea is a type of sleep disorder breathing that causes intermittent pauses in breathing during sleep, leading to transient nocturnal hypoxia and hypercapnia. Obstructive sleep apnoea (OSA) is one of the systemic risk factors for glaucoma which causes irreversible visual field (VF) damage.

1. Introduction

Melatonin is a ubiquitous neurohormone found naturally, both in plants and animals. It is popularly known as sleep inducing hormone or sleep hormone and it is synthesized by pineal gland, to be specific in pinealocytes. Other than pineal gland, it is also synthesized by several peripheral organs including eyes. Ocular structures producing melatonin include retina, ciliary body and lacrimal glands.[1–7] In human body, melatonin is involved in a number of functions but mainly it exhibits circadian rhythm regulation.[8–10] Other functions include regulation of blood pressure, temperature, sleep and immune system regulation. It also has a role in retinal physiology and antioxidant defence modulation.[11–20] In eye, melatonin is synthesized locally and controls the retinomotor movements and protects retinal pigment epithelium from oxidative damage. Glaucoma is a group of disorders consisting of optic nerve damage, associated with visual field changes. This damage is done by progressive multifactorial optic neuropathy resulting in retinal ganglion cell degeneration, causing characteristic optic nerve cupping and hence it leads to irreversible vision loss and blindness.[21] It includes both modifiable (mainly intraocular pressure) and non-modifiable risk factors (age, sex and positive family history). Increased intraocular pressure (IOP) is a major risk factor behind glaucoma. However, there are a number of patients showing visual field deterioration even while having normal IOP which provokes us further to find other associated risk factors e.g. association with other systemic disorders which will causes neurodegeneration.[22]

2. Circadian Variation of Aqueous Dynamics

The rate of aqueous humor turnover is estimated to be 1.0% to 1.5% of the anterior chamber volume per minute, which is 2.4 ± 0.6μl/min (mean ± SD, daytime measurements in adults aged 20–83 years).[20,21] Using fluorophotometry,

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diurnal variations were observed in aqueous humor turnover rates, reflecting a pattern known as the circadian rhythm of aqueous humor flow in humans.[23] Aqueous humor flow is higher in the morning than at night. It is normally about 3.0μl/min in the morning, 2.4μl/min in the afternoon, and drops to 1.5μl/min at night.[24] The mechanism that controls this biologic rhythm is poorly understood. Circulating epinephrine available to the ciliary epithelia may be a major driving force.[25] Epinephrine increases the rate of aqueous flow in sleeping subjects to a greater extent than it did in awake subjects. Timolol reduced the rate in awake individuals, but not in sleeping ones and acetazolamide reduced the rate of flow in both awake and epinephrine-stimulated subjects.[26] Another hypothesis supporting epinephrine influence on circadian rhythm could be ciliary production of this hormone. However, epinephrine concentration in human aqueous humor appears to be very low, ranging from 0 to 0.1 ng/ml.[27] Other hormones, such as melatonin, hormones related to pregnancy, and anti-diuretic hormones, do not appear to alter the normal circadian rhythm of the aqueous flow.

3. Association of Sleep on IOP

Intraocular pressure is a balance between aqueous humor drainage from the eye and it's production. Intraocular pressure is not a constant value rather it varies based on a number of factors. The short-acting influences were food or fluid intake, variations in systemic blood pressure and excessive physical activity. The pattern of variation in the IOP over the course of 24 hours may be related to other diurnal endogenous variations in the body, such as the production of cortisol.[28] The variations in IOP may be due to simple mechanical factors. One such mechanical factor is the tension in the intraocular muscles and the effect they have as they compress the globe during the contraction of ciliary muscles while accommodating. Armaly and Jepson[29] showed that accommodation can reduce IOP. During sleep there will be less accommodative effort than at other times. Another possible factor is alterations in blink pattern, particularly during sleep, which may have influence on the episcleral veins and the collector channels near the limbus. It has been shown that blinking raises the IOP instantaneously and a forceful hard blink may raise the IOP by as much as 50 mmHg. Recent studies have suggested that intraocular pressure is regulated by the adrenal cortex. A relationship has been demonstrated between maximal and minimal levels of diurnal tension measurements and plasma levels of 17-hydroxycorticosteroids. According to Roetth A Jr, osmotic changes in the blood might play a role in diurnal pressure variations through changes in aqueous inflow. However, diet, fasting, rest, or exercises do not alter the tension curve.[30]

The reported mean amplitude of the daily fluctuation ranges from approximately 3-6 mmHg. Amplitude greater than 10 mmHg is generally considered to be pathologic and glaucomatous eyes have been reported to exceed 30 mmHg of diurnal variation. The pattern of the daily cycles has classically been described as having the peak IOP in the morning hours. IOP, Retinal function, Axial length, Pupil size, Palpebral tissue width and Tonic accommodation exhibits diurnal variation. The IOP is maintained at its normal level by balance between the hydrostatic pressure in the capillaries and the difference between the osmotic pressure of the aqueous humor and the capillary plasma.[28]

4. Sleep Disorders

Sleep disorders is one such entity. About 1 out of 4 men and 1 out of 10 women have sleep disorder of one type or the other. Sleep apnoea is one of them which have shown its association with glaucoma in a number of previous studies. It is of 3 types; central, obstructive and mixed. Obstructive sleep apnoea (OSA) involves repetitive collapse of pharyngeal airways while sleeping which results in reduced and shallow breathing temporarily i.e hypopnoea or complete transient cessation. OSA clinically can be defined as 5 events of apnoea & hypopnoea per hour of sleep also known as apnoea- hypopnoea index (AHI). (31) It is graded into 3 categories on the basis of AHI: mild OSA is 5-15 AHI per hour, moderate OSA is 15-30 AHI per hour and severe is >30 events of AHI per hour.[31] Almost 13% of men & 6% of women belonging to the age of 30-70 years, suffer from moderate to severe grades of OSA.[32] Symptomatic prevalence of OSA in men is 22-24% & 9-17% in women.[33,34] Almost 82% of men and 93% of women having moderate-severe grades of OSA still remains to be clinically undiagnosed. OSA presents with excessive and uncontrolled day time sleepiness, persistent irritable mood, memory loss, fatigue and morning headaches.[35–37] Patient's attendant frequently complaints of choking, snoring or cessation of respiration while sleeping which go un-noticed by the patient usually.

Risk factors mainly include obesity and increased body mass index. Others are male gender and several craniofacial abnormalities e.g. tonsil enlargement, uvula and tongue thus hampering the airways.[38–40]

OSA has significant association with life endangering cardiovascular disorders like atrial fibrillation, CAD, arrhythmia and hypertension. Cerebrovascular diseases include raised intraocular pressure, impaired cognitive levels, mood disorders, metabolic disorders including glucose intolerance and lipid profile derangements and also endocrine disorders like diabetes mellitus, hypothyroidism and acromegaly.[40]

OSA is also associated with glaucoma which is the leading cause of blindness worldwide. In OSA patients, the prevalence of open angle glaucoma (OAG) is almost about 2%.[41] According to a study, glaucoma prevalence in OSA is almost 4 times greater than the expected population rate.[42]

Other than glaucoma, OSA is also associated with several ocular disorders e.g. floppy eye lid syndrome, non-arteritic anterior ischemic neuropathy, central serous chorioretinopathy, papilloedema, keratoconus and retinal vascular occlusion.[35]

This article aims to review the postulated association & the proposed pathogenesis involved between glaucoma and OSA.

4.1. Pathophysiology of glaucoma in obstructive sleep apnoea

The proposal of association between OSA and glaucoma has been indicated by numerous studies. It is a popular opinion that OSA can deteriorate or cause glaucoma partly but the involved mechanism is not known exactly so far. Chaitanya et al in his study has reviewed the prexisting theories.[43] They were summarised in three categories majorly i.e. hypoxia, vascular & mechanical factors. All three factors are discussed below in brief.

4.1.1. Hypoxia

Like every other cell in the body, neuronal cells also require oxygen for ATP generation. Oxygen is important for all living cells for cellular function and tissue formation. Especially, neuronal cells need more oxygen and glucose consumption to generate continuous energy in the form of adenosine triphosphate (ATP). Mitochondria in cells need oxygen to breakdown glucose through a process of oxidative phosphorylation to generate ATP. Hence, repetitive apnea–hypopnea episodes or reduction in ventilator drive will cause hypoxia and hypercapnia which will cause decrease in pO2 and an increase in pCO2. These prolonged episodes of hypoxia directly damage the ONH, retinal ganglion cells (RGCs), and its axons. Hypoxia will cause oxidative stress and inflammation by increasing reactive oxygen species and inflammatory markers which subsequently leads to mitochondrial dysfunction of RGCs and glaucoma.[22,44,45]

4.1.2. Vascular factors

OSA causes definite vascular changes and vascular dysregulation of ONH. Studies showed that OSA is associated with hypertension, atherosclerosis of carotid artery, vascular endothelial dysfunction, autonomic dysfunction, and raised intracranial pressure.[22,46]

Retina and optic nerve requires constant blood flow to meet their high metabolic needs. OSA causes insufficient blood supply and nourishment to the RNFL and optic nerve.[47] Carotid artery (internal) through each side of the neck supplies blood to the brain. OSA will cause vascular cell wall changes in carotid artery by building plaques formation inside the wall. It narrows the blood vessel and subsequently may end up with ONH ischemia.[46] Intermittent hypoxia will cause increase in sympathetic nervous system activity which leads to vasoconstriction and systemic hypertension.[48] Raising sympathetic activity during daytime causes autonomic dysfunction. Raise of sympathetic activity and autonomic dysfunction may alter the cerebral and ocular circulation, especially during night as during night, normally sympathetic activity and blood pressure decreases. However, ocular perfusion pressure remains stable because episcleral venous pressure around eye increases during night in supine position. Hence, raise of sympathetic activity and autonomic dysfunction may alter ocular perfusion pressure during night.[22,46] Hypoxia causes oxidative stress, inflammation and decreases the availability of nitric oxide, which is a vasodilator agent and thus damages the endothelium. Hypoxia will increase the levels of endothelin-1 which is a powerful vasoconstrictor. Production of endothelin-1 is also found to be higher in OSA and normal tension glaucoma (NTG) patients, which leads to severe impairment of vasodilator response of blood vessels. Hypoxia-induced endothelin-1 and nitric oxide imbalance in OSA causes vascular dysregulation and affects blood flow to ONH and retina.[22,47,49] It was also found that hypoxia indirectly causes increase in intracranial pressure. It leads to decrease in cerebral perfusion pressure and may disturb the blood flow to ONH, especially during nocturnal systemic hypotension.[44,46,49]

4.2. Prevalence of glaucoma in OSA patients

Walsh and Montclaisir in their study in 1982 have laid emphasis on co-relation between glaucoma and OSA. He diagnosed 5 patients of OSA with glaucoma within the same family.[50] An another researcher Mojon et al did a cross sectional study which included 114 OSA suspects and found that the prevalence of glaucoma was 7.2% in them(5/69). Out of those 5 patients, 2 were diagnosed with normal tension glaucoma (NTG) and 3 had primary open angle glaucoma (POAG). The above study laid emphasis on the fact that both POAG and NTG were associated with OSA. This study paved the way to further studies which tried to establish OSA relation with the above two (POAG & NTG) as independent entities.[42] There was another study by Lin et al who did a retrospective cohort study based on a large population in order to establish the risk of POAG in OSA patients. Total 1012 patients in disease cohort along with randomly selected 6072 controls were taken in this study. All of the cases including the diseased and the control cohorts both were followed up for a period of 5 years in order to look for the development of OAG. The incidence rate per thousand person years of glaucoma was observed to be 11.26 for OSA patients and 6.76 for control cohorts. Hence the results pointed towards the fact that OSA patients were at 1.67 times more risk of developing OAG in the first 5 years of initial diagnosis.[51] Chen et al did another set of retrospective study on Taiwanese population which was based on the available treatment options for OSA

and their associated risk of glaucoma development. They took 2528 patients with OSA and 10112 controls which were selected randomly (age matched). The risk assessment for glaucoma was done for various treatment modalities for OSA e.g CPAP (continuous positive airway pressure) and it was compared to those who did not receive any treatment.[52] Risk of glaucoma development was analysed on the basis of adjusted hazard ratio of glaucoma in OSA after adjusting for age, sex, diabetes mellitus, hypertension and other systemic disorders. The adjusted hazard ratio was found to be 1.88 (95% CI: 1.46- 2.42). Bendel et al in his study included total 100 patients with OSA in order to find the prevalence and association of glaucoma. The glaucoma prevalence was found to be 27%. There was no co-relation between IOP & AHI, as well as between glaucoma & AHI according to this study. Lin et al in another study analysed 209 OSA patients and 38 controls which included normal subjects to find NTG prevalence and risk of OSA severity to develop glaucoma. The prevalence of normal tension glaucoma was found to be 5.7% (12 out of 209). Out of these 12 NTG patients, 1 had mild OSA, 3 had moderate OSA while 8 fell in the category of severe OSA. It showed that with increasing severity of OSA, the prevalence of NTG also increased i.e. in moderate/severe OSA, NTG was 7.1% which was significantly higher than normal/mild OSA. This study basically indicated that the severity of OSA is an independent risk factor for glaucoma development.[53] Another study by Hashim et al focused on analyzing progression and prevalence of glaucoma in treated OSA patients that were followed for a period of 3 years. This study included 39 patients, out of which 12 had moderate OSA while 27 had severe OSA. Prevalence of glaucoma among them was found to be 20.5% (total of 8 patients with 95%CI:9.9%-37%). It inured 7 out of 27 severe OSA patients i.e. 25.9% (at 95%CI:8%-34%) and 1 out of 12 moderate OSA patients (8.3% with 95%CI:0.1%-15%). It established that as severity of OSA increases, the risk of glaucoma development also rises. When they were followed for a period of 3 years and were undergoing treatment simultaneously, it was seen that just 2 patients developed NTG and only 2 patients exhibited further deterioration of glaucoma signifying that proper OSA treatment with good ophthalmic care can control glaucoma. There were several studies which reported relatively lesser prevalence of glaucoma in OSA patients.[54] Geyer et al in his study showed that out of total 228 patients of OSA, only 5 had POAG. Prevalence rate was found to be 2%.[55] This is almost the same prevalence as it was found in general Caucasian population (1.7- 3%). Also, it was supported by Kadyan et al who reported glaucoma prevalence in OSA to be 3.4% which matches to that of normal population (2%). On the whole, above discussed studies indicate towards increased prevalence of glaucoma in OSA and also established significant association with the severity of OSA.[56]

4.3. Prevalence obstructive sleep apnoea in glaucoma patients

Several studies have reported higher prevalence of OSA in already diagnosed glaucoma patients. Mojon et al included 16 caucasians with normal tension glaucoma and observed that 8 out of 16 patients have OSA.[57] The diagnosis of OSA was based on polysomnography. 50% patients were detected with OSA after polysomnography. Out of eight, four had mild OSA; two had moderate OSA while 2 had severe OSA. Blumen Ohana et al carried out a study based on association of OSA with POAG & snoring. 31 patients of POAG who had complaint of snoring underwent polysomnography for the clinical diagnosis of OSA. OSA prevalence was found to be 49% [15/31]. This study indicated that snoring is an important indicator to screen the patients for OSA.[58] Bilgin et al conducted a study to establish association of OSA & NTG. They included 24 already diagnosed cases of NTG & 24 controls which were matched for age. All 48 were put through polysomnography for OSA diagnosis, criterion being AHI ≥20/hour. 41.7% i.e. 10 patients had OSA in NTG group while 12.5% i.e. 3 among controls had OSA thus, significant difference was found between the two groups. Also, the relative risk of developing OSA in NTG group was 3.34 times more as compared to the age matched control group.[59] Various studies are also available which established association of POAG and OSA patients. One such study was conducted by Balbay et al which was based on prevalence of POAG in OSA patients. Total 21 patients diagnosed with POAG were taken and all of them underwent overnight polysomnography evaluation for OSA diagnosis. 7 out of 21 patients has associated OSA i.e. 33.3%. 6 patients (14%) had milder disease while 19.0% had moderate OSA.[60] Another questionnaire based study was conducted by Wang et al to assess quality of sleep in primary angle closure glaucoma (PACG), POAG and among controls by using Pittsburgh sleep quality index. Score of ≥7 was taken as disturbed sleep. 23.62% [47/199] among controls had decreased sleep quality while it was 81.25% & 47.83% in PACG and POAG patients. This shows that PACG patients have relatively more disturbed sleep than that of POAG cases.[61]

4.4. Structural changes in retina & optic nerve head in OSA

4.4.1. Retinal nerve fibre layer

Various studies across the globe have reported relation between RNFL & OSA patients. It was reported that RNFL is thinner in OSA patients as compared to controls. Also, negative co-relation has been found between RNFL thickness & AHI, whereas positive correlation is found between RNFL & O2 saturation in OSA patients. Two

mechanisms have been proposed for RNFL thinning in OSA.

The first mechanism is carried out by series of following events: - As in OSA patients, during sleep there is reduced ventilator drive causing decreased p02 and increased pCO2; resulting hypoxemia leads to increased endothelin production which is a vasoconstricting agent. It hence causes imbalance between vasoconstricting and vasodilatory agents. Hypoxia as a result might lead to loss of ganglion cells and hence RNFL thinning.

The second proposed mechanism is through nocturnal vascular changes caused by OSA's which may cause RNFL thinning. Raised intracranial pressure indirectly through hypoxia during the sleep & hence decreased cerebral perfusion pressure may cause disturbed optic nerve supply in OSA patients.[62–65]

The above explained vascular disturbances can cause diffuse or localised RNFL defects even ahead of actual initiation of glaucoma. These vascular dysregulations along with associated systemic nocturnal hypotension will lead to further damage & RNFL thinning.[42,53] RNFL thinning in OSA patients was first reported by Kargi et al. Thirty-four OSA patients were included in this study which contained 19 mild & 15 severe OSA cases along with 20 controls (age matched). RNFL was measured by GDx scanning laser polarimetry. Significant reduction of RNFL was found in this study as compared to controls. Also, the reduced RNFL thickness has significant co-relation with the disease severity.[56] SLP- enhanced corneal compensation (ECC) and OCT was used by Ferrandez et al. in a cross-sectional study for evaluation of RNFL. Forty OSA patients were included along forty-five controls (age matched). OCT parameters indicated no significant association between controls & OSA patients while SLP-ECC showed reduced superior average RNFL thickness and higher NFI (Nerve fiber indicator) in diseased group than controls.[66] Another study by Sagir et al using 108 OSA patients and controls each showed that thinning of RNFL was present in advanced OSA patients inspite of normal appearing optic disc.[67] Casas et al using 96 OSA patients and 64 controls in observational case control study proved that temporal inner retina had thinner RNFL in severe OSA patients.

4.5. Optic nerve head and macula

A number of studies have focussed on measurements regarding optic nerve head in OSA patients and significant increase in the morphological characteristics of optic nerve head has been found. Also, there are associated macular thickness changes. There is also intracranial vascular dysfunction in OSA which leads to usually mild optic nerve swelling, which further might be the reason for associated increased disc area. As retinal ganglion cells are sensitive to even mild systemic hypoxia, it might be the reason behind increased macular thickness. Hypoxia leads to swollen cell body, disrupted plasma membrane and altered nuclear DNA. Increased macular thickness can be attributed to this swelling, which might lead to atrophy occurring secondarily to neuronal death. This may be the reason behind the fact that mild-moderate grade of OSA's have more macular thickness than that of controls and also severe OSA group.[68–70]

Slit lamp biomicroscopy was used by Tsang et al for measuring cup:disc ratio of optic nerve. 36 patients with moderate and severe OSA combined along with 30 age matched controls were examined. 26.39% had glaucoma suspect discs which was almost 4 times more than that of the controls (6.78%).[68] Casas et al in his study took 50 OSA patients & 33 controls for the assessment of RNFL and ONH assessment using stratus OCT. Results showed that measurements of optic nerve head were significantly higher in OSA patients than the control group. It also showed that severe OSA patients had obvious higher disc area than controls. Moreover, temporal inner macular thickness was found to be higher in mild-moderate variety of OSA i.e. $270\pm12\mu$m when compared to those with the severe OSA patients i.e. $260\pm19\mu$m.[70] The optic nerve head parameters were also studied by Lin et al including 105 OSA patients & 22 age matched controls. No significant difference was found in ONH parameters & macular thickness between the two groups.[70] In an another study by Sergi et al, significant association of AHI with cup disc ratio of bilateral eyes was found.[71]

4.5.1. Choroidal thickness

Choroidal layer of eye is one of the highest vascularised structure of the body. As in OSA, there is imbalance between the NO (vasodilatory agent) & endothelin (vasoconstricting agent) due to raised endothelin levels in blood. Endothelin 1 is amongst one of the major components of choroidal blood flow. It might be the reason behind reduced choroidal thickness and it structural changes. Association of OSA and choroidal thickness was studied by Bayhan et al.[68] Spectral domain OCT was used to measure choroidal thickness in this study which included 92 patients of OSA and 32 controls which were age matched. It was seen that choroidal thickness measurements at 1.5mm & 3mm nasal to the fovea was lesser in the severe OSA group than that of the control group. One of the other finding was that the choroidal thickness at 3mm nasal to the fovea was thinner in patients with severe OSA than those with mild OSA. Xin et al in another study reported that the thickness of choroid when measured subfoveally, was thinner in severe OSA group than the controls, mild and moderate OSA.[69] Kraraca et al stated that there was significant difference between OSA and control groups regarding sub-foveal choroidal thickness. One of the interesting finding of this study was that patients belonging to the OSA group exhibited decreased choroidal thickness

in all areas when compared to the mild, severe and even control group.[72]

4.6. Functional changes of retina in OSA

4.6.1. Visual field change

Ganglion cell death occurring in OSA has various contributing factors which are oxidative stress, hypoxia, vascular dysregulations and compromised optic nerve head perfusion. As a result of the neuronal cell death occurring in OSA, there is RNFL thinning and reduced sensitivity of associated visual field areas. In a study, Ferrandez et al aimed at finding changes in retinal sensitivity in OSA patients by using standard automated perimetry (SAP). It showed significant difference in median deviation of SAP i.e. -1.74±2.8 dB in OSA group and -0.23±0.8 Db in the control group. According to this study, visual field index (VFI) was found to be statistically significant i.e. 97.5% in OSA patients & 99.52 in controls. Also, it was pointed out through this study that AHI has significant co-relation between mean deviation, pattern standard deviation and VFI.[73]

4.6.2. Intraocular pressure

Significant association was found between intraocular pressure with AHI and BMI by a number of studies. It has also been described earlier that increased adipose tissue deposition in the intraorbital area is responsible for patients having high intraocular pressure with BMI in OSA patients, which in turn will lead to raised episcleral venous pressure.[44] Significant IOP levels were found by Lin et al between the two groups i.e. AHI<15 and AHI>15.[74] Moghini et al showed that out of 51 patients with OSA, 7 had raised IOP (>21mm of Hg) and also found significant difference in IOP among the control and OSA group.[75] Adam et al. found no significant difference between the control and OSA group.

4.7. CPAP (continuous positive airway pressure) therapy

The gold standard technique for treating OSA is CPAP which is administered through a nasal or face mask. This therapy prevents upper airway collapse at night. Studies have reported that diurnal variations in IOP are higher in OSA patients and it will increase even more after CPAP therapy. The exact mechanism behind higher diurnal variations in OSA patients and CPAP therapy is unknown. Kiekens et al. measured 24 h IOP for every 2 hr in 21 newly diagnosed OSA patients and after 1 month of CPAP therapy. 24 h IOP fluctuations (difference between trough and peak IOP) of ≥8 mmHg noted in 7 patients at baseline visit and the number increased to 12 patients after 1 month of CPAP therapy. The mean difference between trough and peak IOP was 6.7 ± 1.5 mmHg at baseline and 9.0 ± 2.0 mmHg after CPAP therapy.(79) Another study by Cohen et al. measured 24 h IOP in two different groups. One is OSA with non-CPAP treated group and another one is OSA with CPAP-treated group. The mean IOP of the CPAP and non-CPAP groups measured in sitting position before the sleep period was 13.33 ± 2.04 mmHg and 14.02 ± 2.44 mmHg, respectively (P = 0.9). After 1 min of supine position, IOP increased by 1.93 mmHg and 2.13 mmHg for both the non-CPAP and CPAP groups respectively (P = 0.02, P = 0.001). The IOP increased significantly after 7 h of sleep in the supine position, and the mean IOP of the CPAP and non-CPAP groups was 19.2 ± 5.68 mmHg and 19.69 ± 5.61 mmHg, respectively. Three OSA patients with glaucoma treated with CPAP had mean IOP of 23.75 mmHg after 7 h of sleep.[76]

5. Conclusion

OSA is one of the major risk factors for developing glaucoma. Studies show a strong association between OSA and glaucoma. Complete ophthalmic evaluation should be advised at every follow-up for patients with OSA. Glaucoma patients with obesity and progressive VF damage even under low eye pressure (NTG) should be evaluated for OSA and other sleep disorders. OSA patients being treated with CPAP therapy should undergo regular glaucoma screening and IOP monitoring because CPAP therapy may trigger the glaucoma damage and its progression by raising the IOP.

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None.

7. Conflict of Interest

The authors declare no conflict of interest.

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